Explaining the Aetiology of ADHD by Barkley's Executive Function (Behavioural Inhibition) Model

The mechanism of executive functioning in the human brain has been conceptualized in a myriad of ways over the past decades. Executive function (EF) can be defined as a cognitive control mechanism (Packwood et al., 2011) that helps regulate self-control, inhibit suboptimal behaviour, and generate an appropriate response towards goal-directed behaviour (Dajani et al., 2016). Owing to its varied definitions, deficits in EF have been attributed to define certain psychological disorders, mainly the disorders concerning cognitive and behavioural difficulties such as Autism Spectrum Disorder (Hill, 2004), ADHD (Bekker et al., 2004), Conduct Disorders (Fairchild et al., 2009), Social Anxiety Disorder (Fujii et al., 2013), and so on. Although, there has been ample evidence on the association between EF deficits and ADHD (Antshel et al., 2013), to posit that EF deficits in an individual could be a causation factor for ADHD is somewhat under speculation hypothesis as some of the evidence has stated that EF deficits could be observed only in a minority of ADHD individuals (Barkley & Murphy, 2011). Hence, in an effort to understand EF deficits as a causal mechanism of ADHD, this essay will highlight a theoretical model of the executive function called Barkley's model of Behavioral Inhibition (BI) which could help in understanding the aetiology of ADHD. Furthermore, the essay will showcase the role of social environment and emotional reactivity as a prominent factor in defining ADHD followed by its psychological implications. Also, it will delve into questioning the globally accepted symptomatology of ADHD stating how disrupted attention could be a subtype of 'hyperactivity-impulsivity' along with reflecting on some limitations and future research scope of the proposed theoretical model.

ADHD (Attention-Deficit/Hyperactivity Disorder), broadly characterized as a developmental disorder (Barkley, 1997), has also extended to be categorized as cognitive (Raiker et al., 2012), psychiatric (Drechsler et al., 2020), behavioural (Antshel et al., 2013), and a neurodevelopmental (Dajani et al., 2016) disorder. DSM-5 has defined ADHD as a cognitive disorder with a persistent pattern of inattention, impulsivity, distractibility, and hyperactivity in two or more social settings such as home, school or office (American Psychiatric Association, 2013). The onset of the disorder is marked during childhood with a prevalence rate of 2%-7% (Drechsler et al., 2020) and mostly continues to maintain in teenage and adulthood with 4%-75% of ADHD children maintain ADHD in adulthood, however, with a slight alteration in the initial symptoms (Hervey et al., 2004). Although the definition provides a decent understanding of the symptomatology of ADHD, there are probable chances of ADHD being incorrectly assessed, diagnosed, or treated due to the subjective nature of the symptoms (inattention and impulsivity) (Levelink et al., 2018). Moreover, the manifestation of ADHD symptoms has been witnessed to be heterogeneous which complicates to identification and study of their underlying causal mechanisms distinctly (Martella et al., 2020).

The current understanding of ADHD revolves around three words: inattention, hyperactivity, and impulsivity (aligned with the DSM-5 criteria) (Drechsler et al., 2020). To better comprehend the psychological and behavioural implications of ADHD, it was imperative to introduce a theoretical model which incorporated cognitive-psychological aspects of the disorder. One such model is Barkley's theoretical model of Executive function (EF) or Behavioral Inhibition (BI) (Barkley 1997). BI, categorized as a cognitive process (Raiker et al., 2012) is the ability to restrain an immediate response or emotion to an external stimulus and provide a delay to manifest a more appropriate response depending on the social context (Alderson et al., 2007). The delay in providing an appropriate response is moderated by goal-directed behaviour which is posited as the foundation of Barkley's theory of Behavioral Inhibition (Antshel et al., 2013). According to him, Executive Function in an individual comprises self-control and Behavioural Inhibition (BI) is a requisite to its optimum performance (Barkley 1997; Barkley & Murphy, 2006). He primarily focused on the interplay between the processes of self-control, behavioural inhibition, and executive functioning (Barkley 1997; Antshel et al., 2013) and emphasized the deficits of these functions to define ADHD. For an ADHD individual, Barkley's model postulated that the "deficits in BI" (also called behavioural disinhibition or dysregulation) could be categorized as a hierarchal set of classes or functions which in unison, dysregulated the self-control mechanism of an individual (Badre, 2008). The classes were a) Poor working memory (inability to act on the events), b) Delayed internalization of speech (limited verbal working memory and delayed moral reasoning), c) Immature self-regulation of motivation/affect/arousal (poor social capacity in generating goal-directed behaviour), d) Impaired reconstitution (reduced behavioural and verbal fluency) and finally e) Reduced motor control (impaired execution of goal-directed responses) (Barkley 1997; Hervey et al., 2004). These hierarchical classes of self-dysregulation were attributed to the central underlying deficit of ADHD (Martella et al., 2020). Furthermore, a major portion of the evidential studies has been consistent with the BI theory of ADHD stating behavioural inhibition as a core deficit of the disorder (Crundwell, 2005; Friedman et al., 2003; Rapport et al., 2002). This model was somewhat successful in setting a directly proportional relationship between disinhibition and impaired ability to execute a desired goal-driven behaviour in an ADHD individual.

The BI model for ADHD was designed on the notion that a primary deficit in behavioural inhibition accounted for secondary deficits in executive functions (Bekker et al., 2004). The conceptualization of ADHD as an impaired execution of goal-driven behaviour provided an explorative space to understand the constructs of deficits in optimum arousal (Loo et al., 2009) and emotional reactivity (Retz et al., 2012). Moreover, in terms of emotional dysregulation, the BI theoretical model has been conceptualized as a) Manifestation of greater emotional reaction to events and external stimuli, b) Deficit in being objective to select a desired response concerning the event or situation, c) Withdrawal in assessing social perspective as an outcome of the premature emotional reaction, and finally d) inability to arousal and motivation, with an objective of an optimal goal-directed behaviour (Barkley 1997; Crundwell, 2005). The model also perpetuated that ADHD individuals, as a result of emotional dysregulation showed lower empathy, abnormally high emotional reactivity to provoking

situations, and a deficit in analyzing emotional responses for future situations (Crundwell, 2005). This could possibly indicate that the inability to generate a situational-desired response in a social setting is possibly causing psychological alterations including social animosity at home/school and rising anger and stress levels (Crundwell, 2005; Drechsler et al., 2020), which is not primarily covered under the aetiology of ADHD. Emotional dysregulation in ADHD individuals is also supported by evidential studies, where the individuals with the disorder were characterized by higher emotional reactivity and perception than the control group (Rapport et al., 2002).

Focusing on the secondary deficits of BI i.e., inability to effectively generate arousal and motivation and produce a desired response in a social environment, it can be suggested that emotional and behavioural exchanges in a social set-up constitute an important factor for mental well-being in ADHD individuals (Rösler et al., 2008). These social exchanges between people provide a pathway to regulate adaptive social behaviour and prepare them to manifest a more desirable behaviour for future interactions (Uekermann et al., 2010). The purpose of behavioural inhibition is inherently social (Antshel et al., 2013) and with the BI theory of emotional dysregulation in place, it can be construed that there is a proclivity towards a stronger influence of emotional dysregulation and social circumstances on ADHD individuals. The psychological implications including social withdrawal, peer problems, and family distress generated henceforth can be accounted for the impaired ability to generate desired affect and arousal, deficit in processing social perspective, and inadequacy to manifest a goal-directed response and therefore can be attributed to Barkley's BI theory (Wehmeier et al., 2010).

Despite the efficacy in explaining the majority of the symptoms of ADHD, arguments criticizing the validity and reliability of Barkley's model have resurfaced time and again in the literature (Barkley, 2006; Barkley, 2012; Barkley & Murphy, 2011). The debate has been around two prominent interrelated notions: a) Behavioral dysregulation/disinhibition as the core deficit behind ADHD causation (Retz et al., 2012) and b) BI theory explaining the causal mechanism of inattention in ADHD (Barkley, 2006). Addressing the first limitation, a few studies have claimed that there are no group differences between ADHD and the control group when assessed on emotional dysregulation (Braaten & Rosén, 2000; Crundwell, 2005). This argument could potentially question the validity of Barkley's theoretical model owing to the subjective definitions of executive functioning. The umbrella of executive functions has conveniently added numerous cognitive functions throughout the literature (Marjie Boonstra et al., 2005), and thus could pose a potential drawback to explaining the aetiology of ADHD as the constructs of EF or BI keep developing. Moreover, it has been stated that only a subpopulation of ADHD individuals experiences significant EF deficits (Antshel et al., 2013). This could imply a low ecological validity of the assessments of measuring executive dysfunction (Barkley & Murphy, 2011).

The second criticism of the BI model is that it is incompetent in explaining the behavioural symptom of inattention (Barkley, 2003), which could be perceived as a valid claim as the model perpetuates to describe the aetiology in terms of performance or goal-directed behaviour (Barkley, 2006). This could lead to an implication that the term "inattention" should not be considered as a primary symptom of the disorder. However, this argument is only posed as a drawback here because it challenges the current DSM-5 definition of ADHD. Clinical studies have supported the hypothesis that an attention deficit is not an independent symptom group in ADHD but rather a later developmental symptom of inhibition (hyperactivity-impulsivity) (Barkley, 2006; Retz et al., 2012). To support this argument, a study by Bekker and colleagues (2004) showed that children with the hyperactivity predominant type tend to experience a deficit in attention in their adulthood implying that hyperactivity or behavioural dysregulation transitions into more attention-related difficulties in adulthood (Bekker et al., 2004). The BI model has translated inattention into forgetfulness, lack of time management, and reduced creativity in generating desired/anticipated behaviour for future scenarios (Antshel et al., 2013). Hence, it could be suggested to consider making slight changes or probably omit the terminology of the "inattention" ADHD subtype.

Barkley's BI theory has been instrumental in encouraging individuals to develop and rehabilitate their executive functions and align them to ensure adaptive goal-directed behaviour (Barkley et al., 2011). This could probably be one of the reasons why cognitive-behavioural theory (CBT) has been the most widely used treatment and relatively more successful than other psychological/medical treatments (Weiss et al., 2012). Hence, future research can be dedicated towards a standardized definition of behavioural disinhibition concerning ADHD, redefining the core subtypes of the disorder, and exploring how the BI model can facilitate a more correct diagnosis and treatment of ADHD now when different theories promote effective assessment of the disorder.

The heterogeneity of behavioural and psychosocial symptoms has strengthened the BI model of ADHD. A stronger influence of emotional dysregulation and impaired ability to generate an optimum response in a social setting could possibly account for the psychological understanding of ADHD and has somewhat, broadened the horizon of studying the disorder from a socio-emotional perspective. Barkley's BI model provided a structure by encapsulating the deficits of behavioural inhibition to serve a goal-directed behaviour. This goal-directed or performance-driven approach stresses the thought of considering ADHD as a future concern such that the symptoms could be explained as a result of concern towards manifesting adaptive behaviour in future scenarios and the inability to achieve goals. Bringing ADHD into a social context and as an operationalization of emotional dysregulation has improved our understanding to inculcate behavioural and psychological manifestations (social anxiety, lower empathy, and higher frustration levels) and has created avenues to understand ADHD from a behavioural/psychological perspective. This would not only enhance the etiological trajectory but could also lead to the advancement of psychological treatments that are currently employed to

treat ADHD. Also, witnessing inattention as a subtype of inhibition could aid in a renewed understanding of the disorder where researchers can delve more into exploring the different dimensions of behavioural disinhibition in ADHD individuals. Studies on EF deficits have highly contributed to understanding various underlying mechanisms of psychological disorders and in this case, it has proven to be a relatively trustable tool to understand ADHD through emotional and social constructs.